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CASE REPORT

Ischaemic Sciatic Neuropathy: a Complication of Endovascular Repair of Abdominal Aortic Aneurysm

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Introduction

Endovascular repair of abdominal aortic aneurysm (AAA) is associated with satisfactory short-term results and a reduction in both hospital stay and perioperative morbidity. However, a number of specific complications may occur following AAA stenting including perigraft leak, stent migration, graft-limb dysfunction, post-implantation syndrome and microembolic visceral (renal, colonic) ischaemia.¹

This report describes a patient who developed an ischaemic sciatic neuropathy following AAA stenting, a previously undescribed complication.

Case Report

In March 1998 a 74-year-old male underwent exclusion of a 6.2cm AAA with a right aorto-uni-iliac device (Endovascular Technologies, California, U.S.A.). The distal limb of the stent-graft was deployed in the external iliac artery, the ipsilateral internal iliac artery (IIA) ligated, the contralateral common iliac artery occluded with an Endosoc (Endovascular Technologies, California, U.S.A.) and the left leg revascularised with a femorofemoral crossover. Completion angiography confirmed aneurysm exclusion with patency of the left IIA.

On the first postoperative day the patient complained of pain in his right buttock and of numbness in the right calf and foot. Examination revealed swelling and tenderness of the gluteal muscles, reduced hamstring power, foot drop and a sensory deficit in the calf. Subsequent nerve conduction studies confirmed a high sciatic neuropathy.

At 4-month follow-up motor function had partially recovered but paraesthesia persisted in the leg. The stent-graft and femorofemoral crossover were functioning satisfactorily.

Discussion

Nerve conduction studies confirmed the presence of a high sciatic neuropathy. The coexistent signs of buttock tenderness and swelling suggest that it was ischaemic in origin, since both gluteus maximus and the sciatic nerve are supplied by the inferior gluteal artery, a branch of the IIA. This complication has not been described following endovascular AAA repair, although it has been reported in association with rupture of IIA aneurysms.² The unilateral nature of the lesion and the muscle tenderness excludes spinal cord ischaemia as a cause.

Although the right IIA was ligated it would be expected that the contralateral IIA (patent on angiography) would maintain pelvic blood supply. Thus, in our patient cross-flow was either inadequate or pelvic perfusion was impaired as a result of microemboli release during stent-graft insertion. However, there were no other signs of small vessel occlusion.

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This novel complication of endovascular AAA repair highlights the potential hazards of IIA occlusion. With increasing efforts to offer such therapy to a greater proportion of AAA patients IIA ligation is likely to be performed more frequently than during conventional AAA surgery. This may increase the perioperative morbidity from pelvic ischaemic injury.

References

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